rectly the side upon which the affected tooth is located, but will often point to a perfectly sound tooth as the cause of their pain. A thorough examination should be made, by the aid of the mouth mirror and a fine exploring point, of all suspected teeth. If a tooth is found with a carious cavity of any considerable size, especially if the cavity is sensitive to the touch of an instrument, it is fair to infer that such a tooth is the one giving pain. The diagnosis can be confirmed by the application of a little cold water to the cavity of decay. Where a congested or inflamed pulp exists, this application will cause an exacerbation of the pain.

Having located the tooth which is the seat of the difficulty, its carious cavity should be washed out with a syringeful of warm water, in order to remove irritating particles of food. The next step is to make an application to the exposed pulp, or, if the pulp is not exposed, to the dentine in the neighborhood of the pulp, which will allay the pain.

A simple and efficacious remedy is the oil of cloves. More powerful remedies are: carbolic acid, ninety five per cent.; creosote; a mixture of equal parts of oil of cloves and chloroform; a mixture of equal parts of oil of cloves and creosote.

One drop of any of these remedies is usually sufficient for a single application. The medicine should be applied to the cavity on a pledget of cotton. Care should be taken not to press the cotton too tightly into the cavity, as it might thus become a mechanical irritant to an exposed pulp. In the use of concentrated carbolic acid, care should be taken to prevent its spreading to the adjoining gum and mucous membrane of the lips and cheek.

It is very important, in treating toothache, to know whether the pulp in the affected tooth is alive or dead. If alive, it will respond to thermal changes and be sensitive to exploration in the carious cavity, and should be treated as just described. If the pulp is dead the tooth is usually sore to percussion, and unaffected by applications of cold, though heat will sometimes be painful.

It is not sensitive to the exploration of an instrument in the cavity of decay. The carious cavity of such a tooth should not be plugged with a dressing, but should be opened freely to give vent to the decomposing pulp in the manner described in the section on affections of the peridental membrane.

Closely allied to inflammation of the pulp is abscess of the pulp. This affection, clinically, cannot always be distinguished from the preceding. Upon microscopic examination, however, it is possible to make out true abscess cavities. These may be deeply situated in the body of the pulp, or near its surface.

Among the more advanced pathological changes in the pulp may be mentioned gangrene. This, as in other parts of the body, may be moist or dry. Gangrene follows upon the sudden cutting off of the circulation from the pulp, as a result of acute inflammations, or violence to the tooth of such a kind as to sever the artery at the apical foramen. A gangrenous pulp is of a grayish-green color, of slight consistence, and fetid odor. In such a pulp the normal structural elements become undistinguishable. In dry gangrene the pulp contracts to a very small compass, and the part of the pulp cavity thus left vacant is occupied by a gas of decomposition. Gangrenous pulps, unless the pulp cavity is freely opened, produce severe inflammation of the peridental membrane.

Another group of pathological changes embraces the various forms of calcification to which the pulp is subject. Among such may be mentioned the nodular form. In this variety small nodules of calcareous matter are sprinkled through the substance of the pulp, giving to it a gritty feel. The calcareous matter is similar in chemical composition to dentine, but does not have its characteristic structure. The calcareous nodules are located between the component parts of the pulp, and are not formed at their expense. This condition seems to be compatible with a healthy activity of the pulp, and apparently does not lead to serious consequences. Its etiology has not been explained. Another form of calcification exists, in which the new formation takes the place of the normal tissue of the pulp and is formed at its expense. The calcareous points are found scattered here and there through the pulp, usually in the coronal portion. These points become confluent till an aggregation is formed ranging in size from a grain of sand up to a mass sufficient to fill the entire pulp cavity, coronal and radical portion as well. This form of calcification apparently does not take place when the tooth is in a normal condition, but seems to be induced either by the wearing down of the crowns of the teeth or by caries. In both cases the dentinal fibrils are subject to irritation, and this irritation determines the deposition of lime salts in the substance of the pulp. When once such a deposition begins, it tends to increase till the pulp is changed from a highly sensitive living organism to one practically lifeless, without nerves or vessels, and without the system of tubules which exists in the dentine. During the course of calcification quite severe pain may arise, evidently due to the pressure of the calcareous masses upon the nerve filaments.

A pathological change similar to that occurring in calcification of the pulp is that which takes place in the formation of secondary dentine. This formation is found on the periphery of the pulp at a place adjacent to a carious cavity, and is deposited by the odontoblastic layer of the pulp, which is the formative agent in normal dentine. Secondary dentine is evidently a means taken by nature for the protection of the pulp against the injurious influences incident to advancing caries. Secondary dentine is similar in structure to normal dentine, containing, like it, tubules and fibrils. Its formation is, however, somewhat less regular, and in case the secondary dentine extends far toward the interior of the pulp, it loses its supply of dentinal tubes and becomes less like dentine and more like a calcified pulp. While the formation of secondary dentine in the neighborhood of decay undoubtedly tends for a time to prolong the life of the pulp, experience seems to show that secondary dentine, when once deposited, tends to increase to such proportions as in the end to destroy the life of the pulp.

The process just described is to be distinguished from that deposition of dentine which takes place by degrees during the whole life of the tooth. This deposit is very slow in formation, and takes place uniformly around the inner side of the whole pulp cavity. By this physiological deposition of dentine the pulp cavities in the teeth of old people are reduced to very small proportions. This seems to indicate that the pulp is useful and necessary inversely to the age of the tooth.

There remains to be mentioned a pathological change which increases the size of the pulp. Such an increase can occur only in case the pulp has been exposed. Let such a pulp be subjected to the irritation of foreign substances, and likewise to that of the sharp edges of a carious cavity, and it will sometimes proliferate and fill the cavity.

Why the pulp does not become inflamed and destroyed under such circumstances cannot be explained. This process has usually been noticed in the case of young teeth. The growth may assume the size of a pea, or be larger. It is of fleshy consistence, and is organically united to the pulp by a narrow pedicle, hence it is called polypus of the pulp. In microscopic examination it is found to consist of numerous round and spindle-shaped cells, interspersed with fibrous tissue, and an epithelial covering has been described by some writers. Its blood-vessels pursue a tortuous and irregular course, unlike those in the pulp. No nerves have been found in this tumor, yet it is slightly sensitive to touch, resembling the gum in this respect. Sometimes a muco-purulent discharge issues from its periphery. A polypus protects the pulp against external violence. It is extremely tenacious of life, and will grow again if cut off.

A growth similar to a polypus takes place in some cases of fractured teeth. The pulp, having been exposed,

proliferates through the openings caused by the fracture and forms a tumor outside the pulp cavity. This tumor, morphologically, resembles a true polypus of the pulp; it has, however, a nerve supply, and is quite sensitive to the touch, thus differing from a polypus. Salter has named this growth a "sensitive sprouting of the pulp."

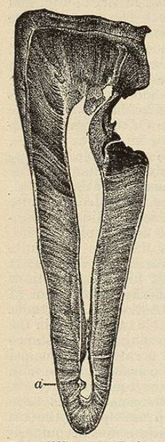

FIG. 4627. — An Incisor Tooth Affected with Caries. *a*, A deposition of secondary dentine about the cavity of decay.

Under pathology of the dentine the most important process to consider is caries. This process affects the enamel and cementum as well as the dentine, but has more to do with the dentine than with the other tissues. In the first place, it may be said that caries of the teeth does not resemble caries of bone. The term caries as applied to the teeth is a misnomer, given at a time when the true nature of the process was not understood. However, the term has become so generally used that it cannot now be easily dropped. The pathological change which occurs in caries is a decalcification and disintegration of the several tissues of the teeth. The latter condition follows very quickly upon the former, on account of the large proportion of earthy constituents existing in the parts attacked. Caries may affect any of the teeth of either dentition, but it affects certain teeth more frequently than others. Magitot has tabulated ten thousand cases of caries occurring in the permanent teeth, and his tables show that the tooth most liable to caries is the first lower molar, after which follow in succession the first upper molar, the second lower molar, first upper bicuspid, second upper bicuspid, upper lateral, second upper molar, upper central, second lower bicuspid, upper canine, first lower bicuspid, upper wisdom, lower wisdom, lower canine, lower central, and lateral. Caries not only shows a preference for certain teeth rather than for others, but it also shows a preference for certain parts of individual teeth rather than for other parts. Those surfaces of the teeth which are smooth and kept clean by the motions of the tongue, lips, and cheeks, are not attacked by caries; while surfaces presenting an uneven contour, abounding in pits and fissures, are its favorite seat. Hence, we find it located in the crowns of the molars and bicuspids, in the pits on the lingual surfaces of the six superior front teeth, and on all approximal surfaces which, though not uneven, are not cleansed by the motions of the mouth. The buccal and labial surfaces of the teeth, just at the margin of the gum, are likewise often the seat of caries. Caries manifests its presence by a change of color in the tissues attacked. This change may be merely from translucency to opacity, or to a variety of colors ranging from yellow to brown, and even black; sometimes a gray or bluish-gray is seen. As a rule, the slower the progress of the disease the deeper the color of the affected parts, and conversely, the more rapid its progress the lighter the color of the affected parts. Caries usually attacks the enamel first, though it may begin with the cementum. It starts in a small pit or fissure, where soon the enamel is found to have lost its peculiarly hard and dense surface. Instead of resisting the most highly tempered steel instruments, as does normal enamel, it crumbles away under slight force. Thus a small opening is made through the enamel to the dentine. This opening may be as large as the head of a pin, or it may be very minute. During this process the enamel has become decalcified and disintegrated. Some authorities say that the centre of the enamel prisms are first affected, and others that the interprismatic substance is first destroyed, in consequence of which the prisms separate and fall to pieces. When once caries has perforated the enamel it no longer confines itself to a narrow area, but spreads out laterally between the enamel and dentine. The degree of lateral extension varies greatly, but seems to be somewhat dependent on the structure of the dentine. If the dentine is well calcified, and with few interglobular spaces, the lateral extension is not so great as when the dentine is imperfectly calcified and abounding in interglobular spaces. The carious process in its lateral extension seems to follow the anastomoses of the dentinal tubules, which are very abundant at the junction of the dentine and enamel. After having affected a certain area on the periphery of the dentine, caries penetrates its substance, following the tubules toward the pulp. Inasmuch as the tubules converge from the periphery of the dentine toward the pulp cavity, the progress of caries is marked by a cone-shaped area, the large end of the cone being in the periphery of the dentine, and the small end pointing toward the pulp. Caries tends to penetrate the pulp cavity, and rarely fails, unless checked by mechanical means. When once the pulp cavity has been penetrated, the pulp is exposed to the degenerative changes already described, and, as a rule, dies and disintegrates. The carious process then invades the pulp cavity, meanwhile spreading laterally through the dentine from the area originally attacked, and disintegrating the enamel from the under side. By degrees the crown of the tooth becomes so hollowed out by the continuous softening and disintegration of the dentine that the shell of enamel left becomes unable to withstand the force of mastication, and consequently is broken away. Nor does caries stop with the destruction of the crown; it continues its work in the root, enlarging the root canal at the expense of the surrounding root substance until the root becomes a mere shell and is finally entirely disintegrated. The carious process in the root is not so rapid as in the crown, and roots may withstand its action for years.

The microscopic examination of the carious process shows the change in color of the affected parts which has been described, and the disintegration of the enamel rods.

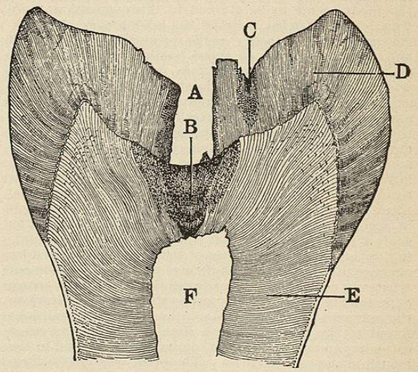

FIG. 4628.—Microscopic Section through a Carious Cavity Occurring in a Molar Tooth. *A*, The initial opening through the enamel; *B*, the cone-shaped area of decay—the affected tissue being discolored and somewhat softened, but not disintegrated; *C*, a minute pit in the enamel where caries has just started; *D*, the enamel; *E*, the dentine; *F*, the pulp cavity.

The tubules of the dentine appear enlarged in calibre; and their size increases as the process advances. The intertubular substance diminishes with the enlargement of

the tubules, and finally disappears with the confluence of adjacent tubules. Micro-organisms are found in great numbers within the tubules.

In the cement, the carious process is similar to that found in the dentine. The lacunæ and canaliculi are enlarged at the expense of the surrounding tissue, which softens and breaks down as the process advances.

Micro-organisms are present as in carious dentine.

A chemical change to be especially noted in connection with all the tissues affected by caries is the acid reaction which is invariably present.

Etiology of Caries.—There are certain predisposing causes upon which all are agreed; of such may be mentioned a faulty calcification of the enamel, which leaves the dentine exposed; a faulty calcification of the dentine, which leaves it less able to resist degenerative changes; a crowded condition of the teeth, on account of which it is difficult to keep the spaces between the teeth clean.

With regard to the exciting or immediate causes of caries, there has been great diversity of opinion. Of the ancient pathologists, some ascribed caries to a disturbance in the "humors of the body." Others regarded it as due to the ravages of worms which infested the oral cavity.

When we come to observers of scientific repute, we find that the older ones held to a vital or inflammatory theory. According to them, the disease began from within, by an inflammatory process of the dentine or pulp, the process in dentine resembling caries of bone; hence the term caries was applied to it also.

The vital theory of caries has been effectually disproved by the fact that when natural teeth have been mounted upon artificial plates, and thus worn in the mouth, they have been subject to caries, precisely resembling the caries of the teeth normally situated in the jaw.

By others, caries was considered to be a sort of gangrene, due to a disturbance in the nutrition of the dentine.

When, however, the secretions of the mouth came to be studied with reference to their possible agency in producing caries, and when they were found to be at times acid, and when, also, the acid fermentations occurring in the mouth came to be studied in this connection, there was developed what is called the acid theory of caries. According to this theory, caries originates from without and not from within, as those holding the vital theory claimed. The active agency in producing it is acids, which are always present in the mouth, due either to acid secretions or acid fermentations. These acids are to a large extent, it is true, neutralized by the alkalinity of the normal mixed saliva; but in some places, as in the crowns of molars and in the spaces between the teeth, the acid secretions are so protected from the neutralizing influence of the saliva that they are able to retain their reaction and attack the enamel, decomposing the phosphate of lime and other mineral constituents, of which it is largely composed. Having penetrated the enamel, the acids act in a similar manner upon the dentine. According to this theory the tissues of the tooth are affected by chemical decomposition, as if there were no vital element whatever concerned. To substantiate this view many experiments were made, by subjecting extracted teeth to the influence of a weak acid solution imitating conditions found in the mouth. Teeth thus treated underwent a softening and decalcification similar to that found in the mouth in the case of caries. The point was thus well established that caries consisted in the decalcification and disintegration of the mineral constituents of the teeth by an acid.

While some have held a vital theory to account for caries, and others an acid theory, still others again have taken middle ground and held a chemico-vital theory.

The discovery of the presence of micro-organisms in the tubules of dentine affected by caries was an important step in advancing our knowledge of the process. The name of leptothrix buccalis was given to these organisms when first discovered. Though their true agency in caries was not at once understood, they were considered to play an important rôle. Extensive investigations have been made to determine more accurately the nature of the micro-organisms found in the mouth, and their relation to the process of caries. The most valuable of these investigations have been conducted by Dr. W. D. Miller, of Berlin. His method has been to infect sterilized culture media of various kinds with neutral saliva or with neutral carious dentine, and he has found, invariably, that, when the culture medium contains sugar, an acid is produced. By successive cultures he has isolated the organisms which produce the acid. Of the organisms he writes as follows: "We have, then, in carious dentine, two distinct fungi—one always, the other often, present; the former surely, the latter probably, producing lactic acid from sugar" ("American System of Dentistry," vol. i., p. 803). Perfectly sound dentine, subjected to a pure culture of the fungi just mentioned in a medium containing sugar, underwent, in course of time, typical caries. According to Dr. Miller—and his theory is now quite generally accepted—the history of caries is as follows: It starts wherever, from the contour of individual teeth or from the relation of one tooth to another, a collection of food is possible. In every such collection are multitudes of micro-organisms which are capable of thriving in the presence of sugar, and of decomposing this substance and forming lactic acid. This acid decalcifies the enamel and forms a small pit which, being constantly filled with food, offers a favorable nidus for the continued growth of the same organisms. When the enamel has been penetrated, the organisms begin to multiply in the tubules of the dentine, and there continue the decomposition of sugar absorbed from the mouth. The resulting lactic acid enlarges the tubules by the decomposition of the mineral constituents of the dentine. It is possible that at one time the secretions of the mouth may be more unfavorable to the life of micro-organisms than at another, since it is well known that, at certain times and in certain individuals, caries progresses very rapidly.

A condition resembling caries, and yet essentially differing from it, is erosion. Erosion is commonly found on the labial surface of the six anterior teeth, either at the margin of the gum, or between it and the cutting edge. It also sometimes affects the bicuspids and molars. Erosion produces shallow cavities, which involve the enamel and penetrate to the dentine. These cavities are larger at their external opening than in their deeper parts, and are smooth, hard, and polished throughout. They present neither the characteristic softening nor undermining growth which are found in caries. The cavities do not rapidly enlarge, but may become of such size as to threaten the life of the pulp. Caries is sometimes superadded to erosion, thus modifying the course of the destructive process. The etiology of erosion is not definitely determined.

Pathological Changes in the Cement.—The most common pathological change of the cement is an hypertrophy, which is due to an irritation of the peridental membrane. This membrane, lying between the cement of the root and the bony alveolus, is at once the formative membrane of the cement of the tooth and of the adjacent bone of the alveolus. When, however, the cement of the root has been completed, the activity of the peridental membrane, so far as its cement-forming function is concerned, normally ceases. It does not resume this function unless subjected to irritation, in which case it may deposit additional cement upon the root in various ways. The deposit may be diffuse, covering the entire root, though most abundant at the apex. It may be nodular, the nodules being found at any point on the root, and being of various sizes; or it may consist of a club-shaped enlargement at the end of the root.

The added cement is similar in structure to the primary cement, the union between the two deposits being, as a rule, not noticeable. In certain cases, however, blood-vessels penetrate this secondary deposit of cement, a condition not found in the primary deposit. Hypertrophy

of the cement has never been observed in the case of the temporary teeth, and affects the permanent teeth during adult life. The teeth most commonly involved are the upper bicuspids and molars, though the others are not exempt. Hypertrophies of the cement are called hypercementoses, osteomata, and exostoses. Of the causes which produce an irritation of the peridental membrane, and consequent hypertrophy of the cement, perhaps the most frequent is caries with its sequelæ, viz., inflammation and death of the pulp, with extension of the inflammation to the peridental membrane. Another cause is the undue pressure which teeth are sometimes subjected to in the process of occlusion and mastication. This arises when many of the teeth have been lost and the few remaining ones are compelled to bear all the strain of service. In such cases the peridental membrane is overworked, literally crowded to the wall, and in consequence may become irritated. The same effect may be produced by the insertion of fillings which project from the crown of a tooth so far as to concentrate the force of occlusion on the filled tooth. While hypertrophy of the cement is commonly due to irritation from undue force, there are cases in which teeth having no antagonists are found to have hypertrophied cement. The symptoms which may arise from an hypertrophy of the cement are caused by the pressure of the new growth upon the nerves of the peridental membrane and upon the nerves of the pulp at the apical foramen. Many cases of hypertrophy exist which occasion no symptoms, the condition becoming known only after extraction. In old people it is usual to find the cement somewhat thickened, and this change can almost be called physiological, so constantly does it occur. Doubtless the process is so gradual that the surrounding tissues accommodate themselves to the enlarged root, and their nerves are subjected to no irritation. In other cases pain is an important and persistent symptom. The pain may be localized and accompanied by a soreness in the socket, or it may be diffused throughout the jaw or reflected to adjoining parts of the head. Severe neuralgias of the head, face, and neck have been found to owe their origin to the hypertrophy of the cement of a tooth. The tooth may appear to be perfectly sound, in which case it is exceedingly difficult to locate the source of the pain. When, however, neuralgias exist in connection with teeth which, though not carious, are the seat of pain or are sore in the socket, it is fair to suspect either an hypertrophy of the cement or a calcification in the pulp. Not only does an enlarged cement cause severe neuralgic pains about the head and face, but cases of epilepsy and insanity have been reported as due to the same cause. A case from Tomes' "Dental Surgery" is in point. "A lad, a farm laborer from Windsor, was admitted into the Middlesex Hospital for epilepsy. The usual remedies were tried for six weeks without effect. His mouth was then examined and the molar teeth of the lower jaw found to be much decayed, the fangs of some alone remaining. Although he did not complain of pain in the teeth or in the jaw, the decayed teeth were removed, and the fangs of each were found to be enlarged and bulbous from exostosis. During the eighteen months that succeeded the removal of the diseased teeth he had not suffered from a single fit, though for many weeks previous to the operation he had had two or three per day."

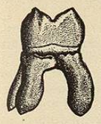

FIG. 4629.—General Hypertrophy of the Cement about the Roots of a Superior Molar Tooth.

A second pathological change of the cement is absorption. This is often found in connection with hypertrophy, and occurs at scattered points and produces depressions in the surface of the cement. In cases of long-continued inflammation about the apex of the root, the cement is likely to be in part absorbed, giving a rough outline to the apex.

Pathology of the Peridental Membrane.—Disease of the peridental membrane may be due to a constitutional disturbance, or to pathological affections of the pulp, or may be dependent upon, or associated with, calcareous deposits upon the root of the tooth. Of the constitutional affections which react upon the peridental membrane, Tomes enumerates rheumatism, syphilis, and the exhibition of mercury.

Inflammation of the peridental membrane from rheumatism is independent of caries, and may involve one or more teeth. The inflammation is distributed over the entire membrane, causing a loosening of the tooth and a soreness in the socket. Its course is subacute or chronic, does not tend to abscess, and is amenable to constitutional treatment.

Inflammation of the peridental membrane from syphilis is chronic. Pus may be discharged around the neck of the tooth, which becomes sore in the socket and loose. If the disease is unchecked the teeth may fall out of their own accord, owing to a complete destruction of the peridental membrane. Associated with this process there often occurs a necrosis of the surrounding bone.

Inflammation from the administration of mercury, whether in the treatment of syphilis or not, is associated with ptyalism, and is of a subacute or chronic character. The teeth become loosened and sore in their sockets, and, if the drug is long continued, a discharge of pus around the neck of the teeth and final loss of the teeth may result.

The effect of phosphorus upon the peridental membrane, though not belonging strictly under constitutional affections, is of great interest. Persons subjected to the fumes of phosphorus, as are those who work in match factories, often have necrosis of the maxillary bones. This necrosis starts with an inflammation of the peridental membrane, which is very sensitive to the irritating fumes of phosphorus. As a result of inflammation the membrane is destroyed, and the bony alveolus being thus cut off in large degree from its source of nourishment, necrosis is invited. It has been found that this disease mainly affects operatives in whose mouth are carious teeth, or who have had teeth extracted while pursuing their occupation. The phosphorus fumes enter a carious cavity and reach the peridental membrane by way of the apical foramen. If, however, the teeth are sound and the gums healthy, phosphorus has little if any destructive effect in the mouth.

Pathological affections of the peridental membrane consequent upon disease of the pulp are of frequent occurrence. When the pulp has become severely inflamed it is common to find, in addition to the symptoms attendant upon simple inflammation of the pulp, a soreness of the tooth in the socket. If the tooth is then percussed with a steel instrument the patient will flinch. This is an indication that the inflammation has proceeded up the root canal and extended to the peridental membrane situated around the apex of the root. Symptoms pointing to inflammation around the root do not always appear during the inflammatory stage of the pulp; they more commonly follow its death and putrefaction. When this has occurred, irritating products of decomposition, both gaseous and liquid, pass up through the canal and set up acute inflammation in the membrane at the apex of the root. In the light of our knowledge of the agency of micro-organisms in inflammatory processes, we must consider that the germs, which were active in producing inflammation of the pulp, are also active in the consequent inflammation of the peridental membrane. This membrane being of connective tissue, and richly supplied with blood-vessels, is an excellent field for inflammatory action, and being closely confined between unyielding walls, and having an abundant nerve supply, is capable of producing symptoms of the severest character. When an inflammatory process has started at the apex of the root, the tissues become swelled and engorged with blood, the condition extending from the apex toward the neck of the tooth. In consequence of the swelling of the membrane, the tooth is pushed slightly from its socket and becomes loose. The clinical symptoms are ushered in by a dull, continuous pain, which is